Both H pylori infection and NSAID use are independent risk factors for the development of Peptic Ulcer disease and associated bleeding

H. Pylori infection is associated with an increased risk of complicated and uncomplicated GD ulcers in NSAID and low-dose ASA users

Cervia II Working Group Report 2006: Guidelines on diagnosis and treatment of *Helicobacter pylori* infection in Italy

Management of *Helicobacter pylori* Infection – the Maastrict IV/Florence Consensus Report - 2012

Helicobacter pylori and NSAIDs

…recommended H pylori eradication before starting long term NSAIDs treatment (>3 months) whilst PPI therapy is advisable in high-risk pts (age > 75 years; personal history of PUD; concomitant therapy with either steroids or anti-coagulant) requiring shorter treatment (level B)

In pts already in long-term treatment with NSAIDs, PPI treatment, misoprostol therapy or H. pylori eradication could be equally chosen (level B)

However, when and ulcer or ulcer complication develops in pts in long-term NSAIDs treatment, H. Pylori infection should be searched and treated and PPI treatment continued (level A).

Eradication reduces the risk of complicated and uncomplicated GD ulcers associated with NSAIDs and low-dose ASA use (level 1B)

H. Pylori eradication is beneficial before starting NSAID treatment. It is mandatory in pts with a peptic ulcer history (level 1B)

H. Pylori eradication does not reduce the incidence of GD ulcers in pts already receiving long-term NSAID treatment. They require continued PPI treatment as well as eradication treatment (level 1B).

Testing for H. pylori should be performed in ASA users with a history of GD ulcer. The long term incidence of PU bleeding is low in these pts after receiving eradication even in the absence of gastroprotective treatment (level 2B)

H. pylori, ASA, NSAIDs

H. pylori, ASA, Glucocorticoids

H. pylori, ASA/NSAID and Glucocorticoids

H. pylori, Coxibs

H. pylori, Clopidogrel or anticoagulants

Curr Pharm Des 2003

COX-2 Inhibition, H. pylori Infection and the Risk of Gastrointestinal Complications

Francis K.L. Chan

Current data on the gastric safety of cyclooxygenase-2 (COX-2) inhibitors in the presence of H. pylori infection are largely derived from animal experiments and indirect clinical evidence. In animal models of H. pylori gastritis, COX-2 inhibitors suppressed prostaglandin synthesis and aggravated mucosal damage. In the human stomach, COX-1 appears to be the predominant source of prostaglandins despite the fact that COX-2 is upregulated in H. pylori gastritis. There are conflicting data on whether H. pylori alters the risk of ulcer in patients receiving COX-2 inhibitors. Among patients with H. pylori infection, rofecoxib reduced the risk of complicated gastric but not duodenal ulcers as compared to naproxen. The advantage of rofecoxib over naproxen also disappeared in patients with H. pylori infection and prior upper gastrointestinal events. In contrast, pooled data suggested that H. pylori increases the risk of ulcer in patients receiving nonselective nonsteroidal anti-inflammatory drugs but not in patients receiving celecoxib. In rodent gastric ulcers, COX-2 was upregulated in the granulation tissue and ulcer margin.

Inhibition of COX-2 delayed healing of experimental gastric ulcer. Limited data showed that COX-2 expression was also increased in human gastric ulcer regardless of the H. pylori status.

The functional significance of COX-2 in human gastric ulcer is unknown.

Clin Biochem 2008: 41:917-9

Role of endogenous cortisol on Helicobacter pylori colonization

Koşan B, et al

Patients with gastric H. pylori colonization have significantly lower cortisol levels when compared with H. pylori negative cases

Br J Clin Pathol 2009;68:251-9

Gastric mucosal injury in systemic lupus erythematosus patients receiving pulse methylprednisolone therapy

Luo JC, et al.

Use of NSAID/ASA, but not H. pylori infection, increases gastric mucosal injury

FEMS Microbiol Lett 2011;318: 68-75

Steroid hormones as bactericidal agents to Helicobacter pylori

Hosada K, et al

Estradiol, androstenedione, and progesteron all have the potential to inhibit the growth of H. pylori

Nephrol Dial Transplant 2011; 2:727-32

High frequency of ulcers, not associated with Helicobacter pylori, in the stomach in the first year after kidney transplantation

Telkes G, et al

Immunosuppressive combinations included: CsA - MMF- GC
CsA - GC
Tacrolimus – MMF - GC

..98% percent of the patients received PPI therapy

H. pylori was found in 20.9% of cases, less than in general and also in uraemic population ($p < 0.0001$)

No association between the presence of H. pylori and ulcers ($p=0.28$)

Steroid pulse treatment for rejection was not associated with more ulcers ($p=0.11$)